

November 30, 1952

Dear Bruce:

While planning some other (general) writing, I've run into the problem of how to refer to your pending manuscript. I had a thought on the matter, on which I would like your consideration. Why do you not issue this paper under your sole authorship, viz. Stocker, 1953, rather than Stocker, Lederberg, and Zinder or Stocker, Zinder and Lederberg, 1953, as previously discussed? You have, of course, my full permission to quote any of my own work that may be needed, and I shall be surprised if ~~Hollomon~~ does not give his assent in similar wise. May I take a similar liberty in quoting the anticipated Stocker, 1953? In addition, it seems to me appropriate that your paper be issued from this department, where most of the work was done. The established form for this address, and for acknowledgment of support (in part) by various agencies appear on the first page of our paper in the Journal of Bacteriology for November. There will be ample opportunity, of course, for your permanent address and for any additional acknowledgments on account of your fellowship.

It turns out that S. abony (Edwards 103) is much more variable in flagellar phase than I had thought, so that this has not been controlled. I shall have to find another system to study the role of the transduces phase in H_1/H_2 determination. However, previous speculations based on the distinctive behavior of FA's of the two phases in transduction to diphasics are still tenable, although somewhat more information is needed on transductions from H_2 . LT-2 seems to be sufficiently stable for these experiments, but it does throw a fair proportion of "masked H's". (As I think of it, H_1 and H_2 seem better symbols for the two major loci for the H antigens. How about F_1 or F_{1a} , 2, ... for the O-form mutations, and P_{af} or the like for the paralyzed?)

The $H_1:F_{1a}$ linkage story is not giving a clear result. The two-step i's mentioned in my previous letter are giving, in different cases, from 50% to 20% i in --x 543. As you will recall, typhimurium gave about 10% i, while the filial and second filial, SW-623, and 623--x543, give more than 90% i. However, I made the mistake of testing other (tymur --x 543) i, in addition to SW-923, and these are giving very diverse ratios. It looks as if different transductions have their own individuality, but the statistics on this are rather troublesome. I shall be surprised if this individuality persists indefinitely, but so far different FA preparations grown on the same host have been consistent. The linkage is the best clue to the finer structure of the genotypic system, whence my detailed interest in it. I have an experiment in the works on differential inactivation of H_1^i in the $H_1^i.F_{1a}$ complex of FA(SW-623); this should be reflected in a shift in the i/b ratio in FA partially inactivated by various mutagens, and may give some insight into the intrinsic determination of this ratio.

Sincerely,

Joshua Lederberg